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UNDERSTANDING THE DISEASE

Driving pressure and transpulmonary pressure: understanding the difference

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The major goal of lung-protective ventilation in patients with acute respiratory distress syndrome (ARDS) is to mitigate the mechanical factors that promote ventilator-induced lung injury (VILI)—including volutrauma (alveolar overdistension) and atelectrauma (cyclic alveolar opening and closing) [1]. Beyond lowering the tidal volume (V_T) and limiting plateau pressure [2], a physiological assessment that estimates stress and strain can help guide ventilator settings. In this context, driving pressure and transpulmonary pressure represent two complementary approaches to quantifying the mechanical constraints applied to the lungs.

Driving pressure

In patients receiving controlled mechanical ventilation without spontaneous breathing efforts, the driving pressure (ΔP) can be measured at the bedside as the difference between plateau pressure (P_{plat}) and total PEEP ($PEEP_{tot}$) (Fig. 1):

$$\Delta P = P_{plat} - PEEP_{tot}. \quad (1)$$

It represents the elastic pressure required to inflate the respiratory system, i.e., the pressure needed to overcome the elastic recoil of the aerated lung and the chest wall when delivering the V_T. ΔP, therefore, equals the V_T divided by the respiratory system compliance (C_{RS}):

$$\Delta P = V_T / C_{RS}. \quad (2)$$

In ARDS, the aerated lung volume is markedly reduced [3], which is why lowering the V_T is a cornerstone of lung-protective ventilation. However, the V_T is generally scaled to the predicted body weight, reflecting the size of the healthy lung, but not the reduced functional lung volume. Because the C_{RS} closely correlates with the end-expiratory lung volume in ARDS, ΔP normalizes the V_T to the functional size of the diseased lung. Therefore, provided that the chest wall compliance is preserved, ΔP expresses the strain imposed on this smaller lung.

In a post-hoc analysis of 3562 patients from nine randomized controlled trials (RCTs), Amato et al. showed that the ΔP was the variable most strongly associated with mortality in ARDS [4]. Overall, a $\Delta P > 14$ cmH_2O was associated with increased mortality. Observational studies have reported a similar threshold [2, 5]. At the bedside, a ΔP exceeding 14 cmH_2O suggests that the V_T is too high for the available lung volume and should prompt a reduction in the V_T, provided gas exchange remains acceptable. This often requires a concurrent increase in the respiratory rate to maintain CO_2 clearance. However, high respiratory rates may themselves contribute to VILI and have been associated with increased mortality in ARDS, although to a lesser extent than elevated ΔP [5]. Whether a low ΔP (*i.e.*, below 10–12 cmH_2O) should prompt an increase in the V_T and a concomitant decrease in the respiratory rate remains an open question. Haudebourg et al. reported that adjusting the V_T to target a ΔP between 12 and 14 cmH_2O was associated with a lower risk of VILI, assessed by the energy delivered by the ventilator to the lungs, compared with a V_T set at 6 ml/kg of predicted body weight [6]. Such a strategy is being evaluated in an ongoing RCT (NCT06322758).

Limitations of the ΔP should be recognized. First, the ΔP conflates lung and chest wall mechanics. Therefore, in patients with markedly reduced chest wall compliance, such as those with elevated intra-abdominal pressure or significant chest deformation, an elevated ΔP may primarily reflect the force required to move the chest wall

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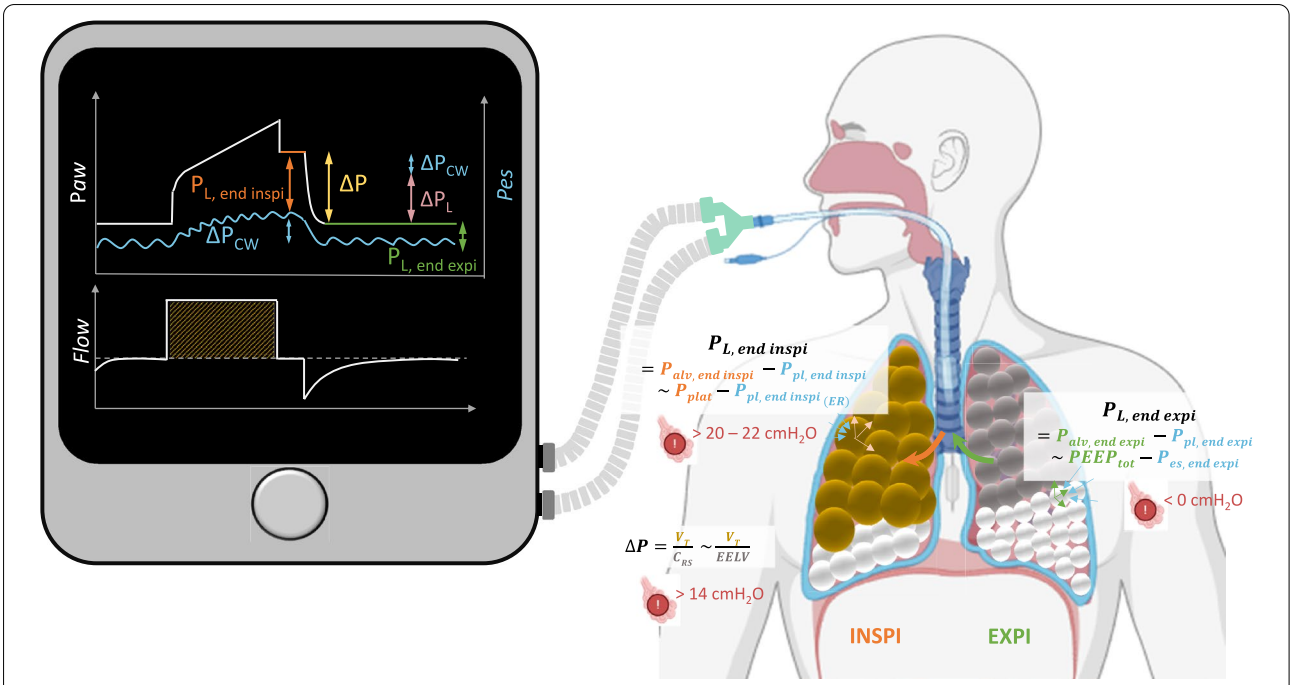

Fig. 1 Driving pressure and transpulmonary pressure: the two sides of the strain. Schematic representation of the respiratory system during volume-controlled ventilation. White units correspond to non-aerated lung regions (collapsed or fluid-filled alveoli). Grey units illustrate the aerated lung volume at end-expiration (EELV), available to receive ventilation. Yellow units represent alveoli distended by the tidal volume at end-inspiration. ΔP respiratory system driving pressure, ΔP_L transpulmonary driving pressure, ΔP_{CW} chest wall driving pressure, $P_{L,end\ inspi}$ end-inspiratory transpulmonary pressure, $P_{alv,end\ inspi}$ end-inspiratory alveolar pressure, $P_{pl,end\ inspi}$ end-inspiratory pleural pressure, P_{plat} plateau pressure, *ER* elastance ratio, V_T tidal volume, C_{RS} respiratory system compliance, *EELV* end-expiratory lung volume, $P_{L,end\ expi}$ end-expiratory transpulmonary pressure, $P_{alv,end\ expi}$ end-expiratory alveolar pressure, $P_{pl,end\ expi}$ end-expiratory pleural pressure, $PEEP_{tot}$ total positive end-expiratory pressure, $P_{es,end\ expi}$ end-expiratory esophageal pressure

rather than lung stress. Second, the ΔP provides no information on the risk of atelectrauma, defined as lung injury caused by cyclic alveolar collapse and reopening during tidal ventilation. Lastly, when the airway opening pressure—defined as the minimal pressure required to open the airways and initiate lung inflation—exceeds the set PEEP, the measured ΔP overestimates its actual value.

Transpulmonary pressure

The transpulmonary pressure (P_L) corresponds to the transmural pressure across the lung, *i.e.,* the difference between airway pressure (P_{aw}) and pleural pressure (P_{pl}). Under static conditions, the P_L equals the transalveolar pressure. The key advantage of the P_L over the ΔP is that it allows one to distinguish lung mechanics from chest wall mechanics. Because P_{pl} cannot be directly measured in clinical practice, it is approximated by esophageal pressure (P_{es}) [7]:

$$P_L = P_{aw} - P_{es}. \quad (3)$$

This direct measurement of the P_L (Eq. 3) adequately estimates the P_L in dependent lung regions but not in non-dependent regions [8].

Another approach to estimate the P_L separates the portion of Paw resulting from lung inflation from that due to chest wall displacement, using the lung-to-respiratory system elastance ratio (E_L/ER_S):

$$P_L = P_{aw} \times E_L/E_{RS}. \quad (4)$$

This approach, which relies on the largely unverified assumption that P_{pl} is zero at functional residual capacity, better approximates the P_L in non-dependent lung regions in experimental studies [8].

Since the P_L is a transmural pressure (the higher the P_L, the greater the distending force on the lung, and therefore the larger the lung volume), limiting the *end-inspiratory* P_L is a logical target to prevent volutrauma. In ARDS, lung elastance represents roughly 70% of respiratory system elastance [9]. Given the recommended plateau pressure limit of 28–30 cmH_2O [10], maintaining *end-inspiratory* P_L below 20–22 cmH_2O using the elastance-ratio method appears reasonable to prevent

overdistension, although clinical evidence remains limited.

Conversely, because negative P_L favors alveolar collapse, maintaining a positive *end-expiratory* P_L with the direct method should help prevent atelectrauma by ensuring alveolar stability during expiration. In a pilot study by Talmor et al. [11], PEEP titrated to achieve *end-expiratory* P_L between 0 and 10 cmH_2O improved oxygenation and showed a trend toward reduced 28-day mortality, although this was not confirmed in the EPVent-2 RCT [12]. Subsequent post hoc analyses of EPVent-2 suggested that the effect of esophageal pressure-guided PEEP may depend on patient severity, and that survival was lowest when *end-expiratory* P_L deviated substantially from values close to 0 cmH_2O, reinforcing the hypothesis that both atelectrauma and overdistension should be mitigated [13]. This approach may still be particularly relevant in obese patients, who often exhibit elevated pleural pressures [9]. In this population, Chen et al. found that an *end-expiratory* $P_L \geq 0$ was associated with improved survival [14]. Notably, targeting a positive *end-expiratory* P_L or an *end-inspiratory* P_L of 20–22 cmH_2O yields different PEEP levels (15). A reasonable approach could be to maintain a positive *end-expiratory* P_L while *avoiding end-inspiratory* P_L values exceeding 20–22 cmH_2O.

The major limitation of P_L is the need for an esophageal balloon catheter, which is invasive and requires specific expertise.

Conclusion

ΔP and P_L provide complementary insights into the mechanical forces contributing to VILI. ΔP enables individualization of V_T according to the size of the "baby lung", whereas P_L offers a framework to personalize PEEP titration, particularly in patients with altered chest wall mechanics or obesity.

Abbreviations

ARDS: Acute respiratory distress syndrome; EELV: End-expiratory lung volume; PEEP: Positive end-expiratory pressure; P_L: Transpulmonary pressure; RCT: Randomized controlled trial; VILI: Ventilator-induced lung injury; V_T: Tidal volume; ΔP: Respiratory system driving pressure.

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Author contributions

GC, AFH and RP were involved in conceptualization and writing. All authors read and approved the final manuscript.

Data availability

Not applicable.

Declarations

Conflicts of interest

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